Adipocyte-specific CD1d-deficiency mitigates diet-induced obesity and insulin resistance in mice

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Supplemental Figure 1. The IFN- γ -STAT1 axis modulates gene expression in 3T3-L1 adipocytes

(a) Phosphorylated stat1 (p-stat1), total stat1 and β -actin (control) in 3T3-L1 adipocytes stimulated by IFN- γ treatment (15 ng/ml) was detected by Western blot. (b) Inhibition of stat1 phosphorylation by ruxolitinib. (c) Modulation of gene expression in 3T3-L1 adipocytes by IFN- γ (30 ng/ml) and ruxolitinib (3 μ M) treatment for 3 days. Ruxolitinib treatment was initiated 1 h before IFN- γ treatment. Representative data from at least 2 independent experiments are shown. Data are shown as mean \pm s.d. Statistical analysis was performed according to the Tukey-Kramer test. *P < 0.05, **P < 0.01.

Supplemental Figure 2. Analysis of CD1d^{f/f}-adipoq-cre mice on SFD

(a) Body weights of CD1d^{ff}-adipoq-cre mice (closed circle) and littermate control mice (opened circle) fed a SFD for 8 wk and weighed weekly (n=4-5 in each group). (b) IPGTT (1 g/kg BW glucose administration) was performed in each group after SFD feeding for 8 wk (n=4-5 in each group). (c) Representative flow cytometric data of iNKT cells ($TCR\beta^+\alpha GC$ tet⁺) and NKT cells ($TCR\beta^+NK1.1^+$) from thymus, spleen, liver and adipose tissue in mice fed SFD at 8 wk of age (n=3-4 in each group). (d) Serum cytokines in CD1d^{ff}-adipoq-cre mice or littermate control mice following α -GalCer treatment (2 μ g/mouse, i.v.) (n=3 in each group). Representative data from at least 2 independent experiments are shown. Data are shown as mean \pm s.d. Statistical analysis was

performed according to the Student's *t*-test. *P < 0.05, **P < 0.01.





